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Food addiction and associated factors in newly diagnosed patients with schizophrenia: a cross-sectional comparison with siblings and healthy controls

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Abstract

Background Despite the potential clinical and treatment relevance of food addiction (FA) among individuals with schizophrenia, the scientific literature on its characteristics and correlates within this population is sparse. Limited knowledge on FA in patients with schizophrenia may impede progress in developing effective treatments for pathological eating patterns and the common obesity and metabolic syndrome problems in this population. Considering these research gaps, the present study aimed to compare the nature and prevalence of FA symptoms among patients with first-episode schizophrenia, their siblings, and healthy controls. As a secondary objective, this study sought to examine the psychopathological correlates of FA in the patients' group.

Methods A cross-sectional study was conducted in Razi Hospital, Tunis, Tunisia, from January to June 2024. A total of 112 newly diagnosed, clinically stabilized patients with first-episode schizophrenia, 77 of their unaffected siblings and 78 healthy controls were included. FA was assessed using the modified version of the Yale Food Addiction Scale (mYFAS 2.0). The Metacognitions Questionnaire (MCQ-30) and the Emotion Regulation Questionnaire were administered to the patients' group.

Results Findings showed a higher prevalence of FA in the patient group (32.1%) compared to both siblings (13.0%) and controls (9.0%). Siblings had higher FA scores compared to controls (16.12 ± 4.95 versus 15.00 ± 6.09; $p < 0.001$). After Bonferroni correction for multiple testing, higher FA scores were significantly associated with less cognitive self-consciousness (Beta = − 0.54), older age (Beta = 0.45), and higher psychological distress (Beta = 0.63).

Conclusion Our findings suggest that people with first-episode schizophrenia are likely to present with co-occurring FA, and experience associated distress. Findings also provide initial support for a possible connection between dysfunctional metacognitive beliefs and FA in patients with schizophrenia, suggesting that cognitive self-consciousness may be a fundamental cognitive process in FA in this population. This may lend some theoretical and clinical implications for alleviating FA symptoms in schizophrenia.

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Plain English summary

There is some evidence to support that food addiction is highly comorbid with schizophrenia. Food addiction might explain the unhealthy eating habits, the poor nutrient intake profile, as well as the high rates of obesity and metabolic syndrome often observed in schizophrenia since the very early stages. However, the co-occurrence of food addiction with schizophrenia remains largely misunderstood and understudied. Such as limited knowledge on the topic is concerning, as it may impede progress in developing effective treatments for pathological eating patterns and addressing the common obesity and metabolic syndrome problems in this population. This study proposed to explore the nature and prevalence of food addiction symptoms in patients with first-episode schizophrenia who have limited exposure to antipsychotic drugs, their siblings, and healthy controls. As a secondary objective, this study sought to examine the psychopathological correlates of food addiction in the patients' group. Findings showed a higher prevalence of food addiction in the patient group relative to both siblings and controls. No significant differences in food addiction severity were found between siblings and controls. Multivariable analyses showed that older age, more psychological distress, and lower "cognitive self-consciousness" metacognitive beliefs were significantly associated with greater food addiction.

Keywords Food addiction, Metacognition, Emotion regulation, Schizophrenia, Disordered eating

Introduction

A large amount of evidence has documented that food has a high addictive potential in humans, and can thus elicit symptoms of substance use disorders [1]. These observations gave rise to the concept of food addiction (FA), or "eating addiction", which commonly refers to abnormal patterns of excessively high consumption of food with high levels of added fats, sugars, sweeteners, or refined carbohydrates, salt and caffeine (such as salty snacks and sweets) [2–4]. To date, there is no fully consensual or universally accepted definition of FA. The condition has largely been defined by mapping the DSM core diagnostic criteria for substance use disorders to eating behaviors [4]. These criteria involve larger amounts consumed than intended, much time spent using or recovering from substance, withdrawal symptoms, tolerance, continual use despite knowledge of consequences, unsuccessful attempts or persistent desire to cut down, and activities given up because of substance use [5]. Of note, FA remains a controversial construct that has invoked debate and prevented its nosographic validation and inclusion in the current diagnostic classification systems (DSM-5 and ICD-11) [5, 6]. The reasons why the concept of FA is criticized are the following: (1) denormalizing and stigmatizing culturally and socially accepted eating behaviors or food preferences can be problematic for mental health specialists; (2) since eating is a physiological behavior, the distinction of pathological aspects of it can be challenging in some circumstances; (3) a high clinical overlap exists between FA and other entities, such as binge eating, bulimia nervosa and obesity; (4) there exists to date no medical clinical/laboratory exams that can diagnose FA, and no evidence-based therapeutic guidelines for its management; and (5) a complete abstinence cannot be a therapeutic objective for people with FA, as it is the case with other addictions [7].

FA appears to include both behavioral dependencies (eating) and dependence on a substance (processed foods with added fats and sweeteners, which have a high addictive potential). However, a systematic review provided evidence that, although both substance- and behavioral-related factors are involved in the addictive process, the construct of FA is more consistent with the diagnostic criteria for substance use disorder diagnoses than behavioral addiction [8]. Indeed, findings from neuroimaging research demonstrated the existence of similarities in neural responses between traditional addiction (such as substance use disorders) and addictive-like eating, especially with respect to their impact on reward pathways of the brain [9, 10]. In addition, some findings support that dopaminergic brain circuits well-established as related to substance use disorders are also incriminated in abnormal eating behaviors, such as overeating [11, 12]. Thus, FA is suggested to be implicated in the obesity pathogenesis via the alteration of the neurocognitive systems implicated in food consumption control [13]. Altogether, there has been a general consistent and converging support among researchers and clinicians for the clinical relevance and validity of FA [14].

FA is reported to occur as commonly as substance use disorders (such as tobacco or alcohol) in the adult general population [14]. A systematic review encompassing 25 studies and 196,211 individuals found an estimated weighted mean prevalence of FA diagnosis of almost 20% [15]. As a recently emerged behavioral pathology, FA is attracting growing attention. Nevertheless, existing studies have focused primarily on some specific populations (predominantly obese, female, with eating disorders [15]), while knowledge on its manifestations and correlates in other clinical populations is still very limited [16]. There is evidence that FA is highly comorbid with other psychiatric disorders [4], with comorbidity (or dual diagnosis)

considered to occur as a rule, rather than an exception in clinical samples [15]. For instance, FA was found more frequently in individuals diagnosed with schizophrenia [17]. Some plausible explanations for this high comorbidity rate have been proposed, including shared genetic background, similar hormonal and personality profiles, common neural and environmental influences, lifestyle, and self-medication [18, 19]. Such comorbidity is not without consequences, as it is shown to result in worse symptoms, lower cognitive functioning, increased risk for physical complications, poorer self-care and overall health, lower treatment adherence, worse treatment outcomes and more health burden for patients [14, 18, 19]. Despite these effects, mental health professionals tend to be more focused on acute psychopathology of a certain disease, and minimize or completely ignore the importance of a comorbid FA that occupies a trivial part of the clinical picture [16]. The present study proposes to delve deeper into this area in the largely understudied population of patients with schizophrenia.

Food addiction in patients with schizophrenia

Patients with psychotic spectrum disorders are more affected by obesity than the general population [20], with an estimated 40–60% of this population being overweight or obese [20, 21]. These findings have also been noted in patients at early stages of the disease. A comparative meta-analysis indicated that both unmedicated and FEP patients (with antipsychotic exposure of less than three months) exhibited an overall rate of metabolic syndrome of approximately 10%, with at least one in five being overweight, having dyslipidemia or high blood pressure [22]. In addition, there is consistent evidence that rates of disordered eating in people with schizophrenia spectrum disorders are two- to threefold higher compared to the general population [23–25]. Patients with psychotic disorders tend to overconsume food both in the forms of increased quantity and frequency, compared to control subjects [25]. A systematic review could identify two studies focusing on FA in patients with schizophrenia who were prescribed antipsychotic medication at chronic stages of illness [23]. The first study by Goluza et al. [17] included a total of 93 outpatients with schizophrenia and found that 26.9% of them met the diagnostic criteria for FA, and that 77.4% of the rest endorsed at least 3 or more symptoms of FA without experiencing associated impairment or distress. The second study by Küçükerdönmez et al. [26] involved 104 outpatients with schizophrenia, and showed that 60.6% had FA. Moreover, patients who met the FA diagnosis criteria were found to eat significantly more carbohydrate, fat, and energy in their diet [26]. At the same time, however, clinicians often tend to miss the presence of disordered eating problems, either due to focusing on psychotic symptoms or because the eating disturbances are not typical or severe enough to meet the full-threshold criteria for eating disorders [23]. Overall, the co-occurrence of FA and schizophrenia remains an under researched area. Besides, although changes in eating behaviors and food preferences were recognized as mechanisms of weight gain in schizophrenia, their related factors are still surprisingly poorly known and studied. Therefore, it appears of utmost importance to explore not only the prevalence, but also related the related factors of FA in patients with schizophrenia.

Possible correlates of food addiction in patients with schizophrenia

Determining potential FA correlates is crucial to understand and consider, as they may help identify patients most at risk for FA and the types of prevention and intervention strategies that could be needed to reduce that risk. Previous research could identify a range of factors related to FA. For instance, a systematic review showed that FA symptom scores were significantly higher in adults aged > 35 years as compared to those younger than 35 years [15]. In addition, previous studies highlighted that dietary preferences are sex-specific [27, 28], which might likely explain the findings that propensity for weight gain is significantly different across sex in patients with schizophrenia spectrum disorder [29]. Other studies reported significant links between disturbed eating behaviors and positive psychotic symptoms medications [30], such as olanzapine and clozapine [31], as well as anxiety and depression [32] in people diagnosed with schizophrenia. Indeed, psychological distress and negative emotional states are known to have a major role in both increasing the risk for addiction [33], and continued intake of high food in spite of harmful consequences [34–36]. A growing body of research findings also suggest that FA is associated with emotion dysregulation in a way that affected individuals are highly aware of negative emotions, but lack the needed skills for coping with them [37]. Finally, strong evidence exists to support that maladaptive metacognitive beliefs are implicated in eating behaviors [38], and were suggested to play a key role in the development and persistence of addictive behaviors [39]. Metacognition can be defined as the ability to reflect on, assess, and control cognitive processes such as perception, memory, and decision-making [40]. Notably, metacognitive beliefs seem to be implicated differently in restrictive versus unrestricted/binge-spectrum eating behaviors [38].

Rationale and aim of the study

As previously posited by Goluza et al. [17], the investigation of FA in patients with schizophrenia may explain the unhealthy eating habits and the poor nutrient intake profile often observed in schizophrenia. Such data is essential to help advance our understanding of the factors contributing to the increased susceptibility to weight gain and obesity in this population [41]. This field of investigation is especially relevant when considering patients at early stages of the disease, as preventing obesity and cardiometabolic risk at more advanced stages can be more difficult to achieve [22]. However, no previous research could be identified that investigated FA in early course patients with schizophrenia, with the very few existing studies having been performed among patients with chronic schizophrenia and established on long-term antipsychotic medication. Studies focusing on chronic patients are subject to many confounding variables such as long-term exposure antipsychotic drugs or poly-medication, more severe symptoms, accumulating relapses and hospitalizations, as well as higher rates of comorbidities. Furthermore, the presence of a first-degree relative of one proband with schizophrenia is considered one of the most important risk factors for schizophrenia, as it carries an estimated eight-fold higher odds ratio of developing the disease [42]. Thus, research of patients' siblings who have a high genetic load for schizophrenia could advance our understanding of clinical markers predicting the onset of the disease. To our knowledge, no studies have examined FA in healthy young people with a high genetic risk for schizophrenia. Considering these research gaps, the present study aimed to investigate the nature and prevalence of FA symptoms in patients with first-episode schizophrenia, their siblings, and healthy controls. As a secondary objective, this study sought to examine the psychopathological correlates of FA in the patients' group. It is expected that patients and their young healthy siblings will exhibit similar rates of FA, which would be higher than those displayed by controls. It is also anticipated that patients with more severe FA symptoms will have more distress, lower functioning, greater psychotic symptoms, more emotion dysregulation and more maladaptive metacognitive beliefs.

Methods

Participants and procedure

A cross-sectional study was conducted in Razi Hospital, Tunis, Tunisia, from January to June 2024. The target population consisted of three groups: a group of patients with schizophrenia, a group of their non-affected siblings, and a group of healthy controls. Data gathering was performed by the same interviewer (i.e., one of the authors [YB]), and lasted between 15 and 30 min for each individual. Assessments of patients and their siblings were all carried-out at the department of psychiatry, using a traditional paper-and-pencil technique. Each participant was informed that they are free to accept or decline to take part in the research, or withdraw at any time without penalty. Patients and siblings were also informed that participation would not impact patients' further care. Before starting the interviews, all participants were assured of the confidentiality of their personal data and responses, and written informed consent was obtained from each patient.

Inclusion criteria for the patients group included the following: (1) being aged 18 to 40 years; (2) being an outpatient diagnosed with a first episode of schizophrenia by at least two psychiatrists through independent clinical interviews according to the DSM-5 criteria [5]; (3) having less than three months of treatment with an antipsychotic medication [43]; (4) being clinically stable, which was defined as follows: "be symptomatically stable, as judged by the treating physician, be receiving a stable dose of an antipsychotic drug for at least 4 weeks before the survey and be in good general physical health" [44]; and (5) having at least one eligible sibling who can participate in our research. After having patients' permission, siblings were approached and invited to participate if they were aged 18–40 years, and did not have a lifetime or current diagnosis of a psychiatric disorder as per SCID-CV [45]. Finally, the control group consisted of individuals aged 18–40 years, who did not have a family history of psychotic disorders, and did not have a lifetime or current diagnosis of a psychiatric disorder as per SCID-CV [45]. Control subjects were recruited from hospital staff and persons from the community on a voluntary basis. Participants were excluded if they did not complete the questionnaire. After excluding seven patients, nine siblings and fifteen controls for incomplete answers, the final sample was composed of 112 newly diagnosed, clinically stabilized patients with schizophrenia, 77 of their siblings and 78 healthy controls.

Measurements

Socio-demographic information gathered included the following: sex, age, occupation, marital status, family monthly income, living area, living situation, physical exercise, personal history of any medical condition, personal history of suicide attempts and/or self-harm, substance use (tobacco, alcohol, cannabis, other drugs). Besides, clinical variables collected in the patient group involved the following: antipsychotics and other medication use, age at onset of the disease, and duration of untreated psychosis (in months). The three groups (patients, siblings and controls) were administered a self-report questionnaire to collect information on

sociodemographic and other study variables using the following four self-report measures: the Emotion Regulation Questionnaire (ERQ), the 30-item Metacognitions Questionnaire (MCQ-30), the Depression, Anxiety, Stress Scales (DASS-8), and the modified version of the Yale Food Addiction Scale (mYFAS 2.0). In addition, patients were administered semi-structured interviews using two interviewer-rated assessments: The Global Assessment of Functioning (GAF) and The Positive and Negative Syndrome Scale (PANSS).

The global assessment of functioning (GAF)

The GAF [46] is an interviewer-rated assessment of global functioning. It is used to assess levels of psychological, social, and occupational functioning of each participant at the present time. It consists of a numerical scale from 0 to 100. The GAF is divided into 10 levels that reflect varying degrees of functioning based on two components: symptoms' severity and function.

The Positive and Negative Syndrome Scale (PANSS)

The PANSS is an interviewer-rated scale used to measure the severity of symptoms in patients with schizophrenia [47]. The PANSS is composed of 30 items divided into three distinct dimensions (i.e., positive scale, negative scale and general psychopathological scale). Each item can be scored on a seven-point Likert-type scale ranging from 1 (absent) to 7 (extreme). Higher total PANSS scores reflect more severe symptoms [48] (Cronbach's $\alpha = 0.85$).

The modified version of the Yale Food Addiction Scale (mYFAS 2.0)

The mYFAS 2.0 [49] is an updated version of the original 25-item YFAS [50]. This scale is self-reported, used to assess FA symptoms, and is not intended to be a diagnostic tool. It contains nine items; seven items refer to the seven DSM diagnostic criteria for substance use disorders (e.g., "I have had physical withdrawal symptoms such as agitation and anxiety when I cut down on certain foods") and two items assess clinical significance (e.g., "My behavior with respect to food and eating causes significant distress"). Items are scored on an a five-point scale ranging from 1 (never) to 5 (4 or more times a week or daily). A respondent who has at least 3 of the 7 dependence symptoms and meets the criterion for clinical significance is considered as having a FA status [49, 51]. The Arabic version of the scale was used [52], which yielded a Cronbach alpha value of 0.88 in the present sample.

The depression, anxiety, stress scales (DASS-8)

The DASS-8 [53] is an Arabic-language shortened version of the DASS-21 [54]. It is a self-report scale that contains only eight items divided into three dimensions, i.e. anxiety (3 items), depression (3 items), and stress (2 items). Each item should be rated on a four-point scale from 0 (did not apply to me at all) to 3 (applied to me very much or most of the time). Only total scores were considered in this study. Higher total scores signify greater psychological distress [55] (Cronbach's $\alpha = 0.82$).

The 30-item Metacognition Questionnaire (MCQ-30)

This self-administered scale assesses maladaptive metacognitive beliefs through 30 items and the five following dimensions: Cognitive self-consciousness, Positive beliefs about worry, Negative beliefs, (Lack of) Cognitive confidence, and Need to control thoughts [56]. Items are scored on a 4-point Likert-type scale ranging from 1 (Disagree) to 4 (Agree). The Arabic validated version of the MCQ-30 was adopted [57], with the following Cronbach's α values: cognitive confidence ($\alpha = 0.85$), positive beliefs ($\alpha = 0.85$), cognitive self-consciousness ($\alpha = 0.82$), negative beliefs ($\alpha = 0.90$) and need to control thoughts ($\alpha = 0.83$).

The Emotion Regulation Questionnaire (ERQ)

The ERQ [58] is a self-report measure composed of 10 items and aimed at assessing maladaptive emotion regulation strategies through two dimensions: (1) Cognitive Reappraisal, which is considered a positive emotion regulation strategy associated with positive clinical outcomes (e.g. "I control my emotions by changing the way I think about the situation I'm in") and (2) Expressive Suppression, which is considered a negative emotion regulation strategy associated with poor clinical outcomes and wellbeing (e.g., "I control my emotions by not expressing them"). Items are rated on a 7-point Likert scale from 1 (Strongly Disagree) to 7 (Strongly agree). The Arabic validated version of the ERQ was used [59], with a Cronbach's alpha values of 0.84 for the expressive suppression subscale and 0.73 for the cognitive reappraisal subscale.

Statistical analysis

Data analysis was done using the SPSS software v.27. The skewness and kurtosis values of the FA score varied between − 1 and + 1 showing normal distribution. Consequently, the LOG transformation was applied, which later showed normal distribution. The Student's t test was used to compare two means, the ANOVA test to compare three or more means and the Pearson correlation test to correlate two continuous variables. Bonferroni multiple testing correction was applied; the new p value = 0.002 was calculated by dividing 0.05 by the total

Table 1 Characteristics of the study sample (N = 267)

	Patients (N = 112) Mean ± SD	Siblings (N = 77)	Controls (N = 78)	*p*
Age (years)	24.44 ± 5.41	25.21 ± 6.55	26.99 ± 3.63	**0.005**
	N (%)			
Sex				**0.001**
Male	90 (80.4%)	43 (55.8%)	52 (66.7%)	
Female	22 (19.6%)	34 (44.2%)	26 (33.3%)	
Occupation				**< 0.001**
Unemployed	35 (31.3%)	7 (9.1%)	2 (2.6%)	
Student	25 (22.3%)	40 (51.9%)	36 (46.2%)	
Part time	40 (35.7%)	6 (7.8%)	0 (0%)	
Full time	12 (10.7%)	24 (31.2%)	40 (51.3%)	
Marital status				0.234
Single	102 (91.1%)	66 (85.7%)	73 (93.6%)	
Married	10 (8.9%)	11 (14.3%)	5 (6.4%)	
Family monthly income (Tunisian Dinar)				**< 0.001**
< 500	80 (71.4%)	0 (0%)	7 (9.0%)	
500–1500	29 (25.9%)	26 (33.8%)	11 (14.1%)	
> 1500	3 (2.7%)	51 (66.2%)	60 (76.9%)	
Living area				**0.009**
Rural	25 (22.3%)	10 (13.0%)	5 (6.4%)	
Urban	87 (77.7%)	67 (87.0%)	73 (93.6%)	
Living situation				0.435
Parents	88 (78.6%)	61 (79.2%)	53 (67.9%)	
Friends	12 (10.7%)	8 (10.4%)	11 (14.1%)	
Alone	12 (10.7%)	8 (10.4%)	14 (17.9%)	
Physical exercise				0.884
No	62 (55.4%)	44 (57.1%)	46 (59.0%)	
Yes	50 (44.6%)	33 (42.9%)	32 (41.0%)	
Personal history of any medical condition				0.213
No	98 (87.5%)	71 (92.2%)	74 (94.9%)	
Yes	14 (12.5%)	6 (7.8%)	4 (5.1%)	
Personal history of suicidal attempts				**< 0.001**
No	78 (69.6%)	75 (97.4%)	75 (96.2%)	
Yes	34 (30.4%)	2 (2.6%)	3 (3.8%)	
Personal history of self-harm				**< 0.001**
No	65 (58.0%)	70 (90.9%)	71 (91.0%)	
Yes	47 (42.0%)	7 (9.1%)	7 (9.0%)	
Smoking				**< 0.001**
No	32 (28.6%)	44 (57.1%)	34 (43.6%)	
Yes	80 (71.4%)	33 (42.9%)	44 (56.4%)	
Alcohol drinking				0.625
No	69 (61.6%)	42 (54.5%)	46 (59.0%)	
Yes	43 (38.4%)	35 (45.5%)	32 (41.0%)	
Cannabis use				**< 0.001**
No	63 (56.3%)	60 (77.9%)	63 (80.8%)	
Yes	49 (43.8%)	17 (22.1%)	15 (19.2%)	
Other illegal drug use				**0.004**
No	95 (84.8%)	74 (96.1%)	75 (96.2%)	
Yes	17 (15.2%)	3 (3.9%)	3 (3.8%)	

Numbers in bold indicate significant p values

Table 2 Endorsement of each symptom of the Yale food addiction scale and food addiction scores by study group

	Patients (N = 112)	Siblings (N = 77)	Controls (N = 78)	*p*	Effect size
Symptom, N (%)					
1. Food taken in larger amount and for longer period than intended	14 (12.5%)	8 (10.4%)	4 (5.1%)	0.235	0.104
2. Persistent desire or repeated unsuccessful attempt to quit certain foods	14 (12.5%)	1 (1.3%)	1 (1.3%)	**< 0.001**	0.233
3. Much time/activity to obtain, use, recover	47 (42.0%)	23 (29.9%)	6 (7.7%)	**< 0.001**	0.316
4. Important occupational, social, or recreational activities reduced or given up	42 (37.5%)	10 (13.0%)	12 (15.4%)	**< 0.001**	0.270
5. Characteristic withdrawal symptoms when cutting down on certain foods; substance taken to relieve withdrawal	39 (34.8%)	3 (3.9%)	4 (5.1%)	**< 0.001**	0.396
6. Consumption of the same types or amounts of food continues despite knowledge of adverse consequences	76 (67.9%)	42 (54.5%)	41 (52.6%)	0.061	0.145
7. Tolerance (marked increase in amount; marked decrease in effect)	72 (64.3%)	36 (46.8%)	39 (50.0%)	**0.033**	0.160
Clinical significance, N (%)					
8. Significant distress	31 (27.7%)	13 (16.9%)	11 (14.1%)	**0.047**	0.151
9. Impaired functioning	28 (25.0%)	13 (16.9%)	15 (19.2%)	0.365	0.087
Food addiction Total scores by using the mYFAS, Mean ± SD	21.44 ± 9.56	16.12 ± 4.95	15.00 ± 6.09	**< 0.001**	0.134
Food addiction status (% yes)[a]	36 (32.1%)	10 (13.0%)	7 (9.0%)	**< 0.001**	0.265

[a] Food addiction status was based on presenting at least 3 of the 7 dependence symptoms and meets the criterion for clinical significance [51]; Numbers in bold indicate significant p values

number of variables tested (=31). A linear regression was conducted using the FA score as the dependent variable and all factors that showed a significant p value after Bonferroni correction ($p < 0.002$) in the bivariate analysis as independent variables. $P < 0.05$ was deemed statistically significant in the final model.

Results

A total of 112 patients, 77 siblings and 78 controls were enrolled in the study. All characteristics of the sample are summarized in Table 1. Besides, clinical and psychological data of the patients' group is displayed in Table S1 (Supplementary material).

Food addiction among patients, siblings and controls

A higher mean FA score was found in patients relative to siblings and controls, with a significant difference between patients and siblings ($p < 0.001$) and between patients and controls ($p < 0.001$) (Table 2). The most common FA symptoms endorsed at least "two to three times per week" in the patient group were tolerance and continued overeating despite psychological or physical problems (2.48 ± 1.43), continued use despite knowledge of adverse consequences (2.52 ± 1.50), and much time/activity to obtain, use, recover (2.47 ± 1.22).

Bivariate analysis of factors associated with food addiction

Findings of bivariate analysis of factors associated with food addiction are presented in Tables 3 and 4. Older age ($r = 0.31$) and higher psychological distress ($r = 0.36$) were significantly associated with more FA, whereas higher cognitive reappraisal ($r = -0.32$) and cognitive self-consciousness ($r = -0.32$) were significantly associated with less FA (Table 4).

Multivariable analyses

Higher cognitive self-consciousness (Beta = − 0.54) was significantly associated with less FA, whereas older age (Beta = 0.45) and higher psychological distress (Beta = 0.63) were significantly associated with more FA (Table 5).

Discussion

Limited knowledge on FA in patients with schizophrenia is concerning, as it may impede progress in developing effective treatments for pathological eating patterns and addressing the common obesity and metabolic syndrome problems in this population [20, 21]. To the best of our knowledge, this is the first study to investigate the nature and correlates of FA in young patients with early-stage schizophrenia who have limited exposure to antipsychotic drugs, and the first to investigate FA symptoms in patients, their unaffected siblings and control subjects. Findings showed a higher prevalence of FA in the patient group relative to both siblings and controls. No significant differences in FA severity were found between siblings and controls. Multivariable analyses showed that older age, more psychological distress, and lower "cognitive self-consciousness" metacognitive beliefs were significantly associated with greater FA.

Table 3 Bivariate analysis of factors associated with food addiction among patients (n = 112)

	Mean ± SD	*t* / *F*	*p*	Cohen's d
Sex		0.065	0.948	0.015
Male	21.47 ± 9.58			
Female	21.32 ± 9.70			
Occupation		0.226	0.878	0.006
Unemployed	21.71 ± 10.80			
Student	20.76 ± 10.11			
Part time	21.05 ± 8.13			
Full time	23.33 ± 9.92			
Marital status		0.220	0.826	0.073
Single	21.50 ± 9.72			
Married	20.80 ± 8.12			
Monthly income (Tunisian Dinar)		0.128	0.880	0.002
< 500	21.54 ± 9.97			
501–1500	21.45 ± 8.50			
> 1500	18.67 ± 11.24			
Living area		0.287	0.287	0.243
Rural	23.24 ± 10.27			
Urban	20.92 ± 9.34			
Living situation		0.078	0.925	0.001
Parents	21.63 ± 9.43			
Friends	20.75 ± 9.72			
Alone	20.75 ± 11.09			
Physical exercise		1.72	0.088	0.327
No	22.82 ± 10.23			
Yes	19.72 ± 8.44			
Personal history of any medical condition		− 0.92	0.358	0.263
No	21.12 ± 9.55			
Yes	23.64 ± 9.68			
Personal history of suicidal attempts		− 0.47	0.636	0.097
No	21.15 ± 9.27			
Yes	22.09 ± 10.30			
Self-harm		− 1.56	0.123	0.307
No	20.22 ± 8.72			
Yes	23.13 ± 10.47			
Smoking		− 0.33	0.744	0.068
No	20.97 ± 9.88			
Yes	21.63 ± 9.48			
Alcohol drinking		0.16	0.875	0.031
No	21.55 ± 9.46			
Yes	21.26 ± 9.82			
Cannabis use		0.78	0.434	0.149
No	22.06 ± 9.56			
Yes	20.63 ± 9.60			
Other illegal drug use		1.17	0.244	0.308
No	21.88 ± 9.53			
Yes	18.94 ± 9.61			
Antipsychotics use		0.62	0.541	0.011
First generation	20.48 ± 9.62			
Second generation	22.26 ± 9.11			

Table 3 (continued)

	Mean ± SD	*t* / *F*	*p*	Cohen's d
First and second generations	19.29 ± 13.59			
Mood stabilizers		0.57	0.569	0.291
No	21.54 ± 9.54			
Yes	18.75 ± 11.32			
Benzodiazepines		0.27	0.787	0.051
No	21.71 ± 8.92			
Yes	21.21 ± 10.13			
Anticholinergics		0.55	0.585	0.104
No	21.90 ± 9.76			
Yes	20.90 ± 9.38			

The prevalence of FA in patients was of 32.1%. This finding is in agreement with the two previous studies available, which reported higher prevalence rates of FA in chronic outpatients with schizophrenia compared to those observed in the general population, ranging from 26.9% [17] to 60.6% [26]. A systematic review found a weighted mean prevalence of FA across 25 studies and 196,211 participant of almost 20%, whereas rates were significantly lower in non-clinical populations (approximately 16%) [15]. Engaging in abnormal patterns of increased eating of food, such as binge eating or night eating, were found to be highly common in the schizophrenia population, with prevalence rates up to 20%, that is around 5 times higher than in the general population [60, 61]. To explain these rates, as well as the fast-food and sweet cravings seen in schizophrenia [27, 62], it has been suggested that dopamine dysfunction and dysfunctional reward processing in the brain play a pivotal role in FA among patients with schizophrenia [17, 26]. Other hypothesized mechanisms include an altered dopamine/acetylcholine ratio in some areas of the brain (e.g., the nucleus accumbens) [63], or dysregulation of serotonin neurotransmission [64]. Although siblings had higher rates of FA than controls (13.0% versus 9.0%, respectively), the difference did not reach statistical significance, which might be due to the relatively small number of siblings. Future comparative case–control studies using larger samples are needed to explore these questions further.

Beyond clinical manifestation and prevalence of FA in patients with first-episode schizophrenia, the current research sought to identify related psychological and social factors. It was observed that FA scores had a close relationship with psychological distress. Several studies described an important overlap between addictive-like food consumption and psychological distress, including stress, depression and anxiety symptomatology [65–67]. In this regard, changes of food preferences and higher consumption of palatable foods have been suggested as a coping strategy that helps mitigate distress [68]. This finding offers additional support for the clinical utility and relevance in clinical practice of FA, particularly in regard to potential distress, and suggest that people with emerging psychosis may experience clinically significant levels of FA, which could contribute to increased distress from the early stages of the disease.

Our results showed that, among the various metacognitive processes, only one remained significantly and negatively associated with FA in the in the final multivariable analysis model, that is, cognitive self-consciousness. These findings are in line with previous literature observing that the less the tendency to monitor one's thought, the less the tendency to restrict one's food intake [69]. Cognitive self-consciousness refers to individual's preoccupation with, and self-monitoring of own thoughts (e.g., 'I constantly examine my thoughts'). This metacognitive domain is conceptualized to play a role in awareness of other maladaptive metacognitive beliefs; this persistent monitoring of thoughts can, in turn, lead to a sense of uncontrollability over intrusive thoughts [70, 71]. According to our findings, we may imply that patients with less cognitive self-consciousness may exhibit greater levels of FA symptoms. The relationship between cognitive self-consciousness and FA is challenging to interpret due to the cross-sectional design and the uncertainty regarding the direction of causality. However, it could be suggested that a person who is not fully preoccupied with their own thoughts may be less self-critical about their body and excessive eating behaviors [72]. This study's results provide initial support for a possible connection between dysfunctional metacognitive beliefs and FA in patients with schizophrenia, suggesting that cognitive self-consciousness may be a fundamental cognitive process in FA in this population. This may lend some theoretical

Table 4 Pearson correlation matrix of continuous variables

	1	2	3	4	5	6	7	8	9	10	11	12	13
1. Food addiction	1												
2. Age	0.31**	1											
3. Age at onset of schizophrenia	0.28**	0.96***	1										
4. Duration of untreated psychosis	0.22*	0.46***	0.21*	1									
5. Functioning	− 0.20*	− 0.09	0.04	− 0.44***	1								
6. PANSS total	0.12	0.21*	0.09	0.47***	− 0.49***	1							
7. Psychological distress	0.36***	0.04	0.04	0.03	0.07	0.05	1						
8. Cognitive reappraisal	− 0.32***	− 0.10	− 0.10	− 0.05	0.08	0.02	− 0.14	1					
9. Emotional suppression	− 0.13	− 0.05	− 0.06	0.02	− 0.13	0.06	0.12	0.67***	1				
10. Cognitive confidence	− 0.07	− 0.02	− 0.01	− 0.04	0.15	− 0.01	0.40***	0.37***	0.47***	1			
11. Positive beliefs	− 0.01	− 0.07	− 0.05	− 0.13	0.22*	− 0.25**	0.39***	0.19*	0.27**	0.42***	1		
12. Cognitive self-consciousness	− 0.29**	− 0.09	− 0.06	− 0.13	0.04	0.03	0.10	0.41***	0.30**	0.12	0.07	1	
13. Negative beliefs	0.04	− 0.03	0.01	− 0.09	0.03	− 0.002	0.60***	0.10	0.38***	0.45***	0.45***	0.19	1
14. Need to control thoughts	− 0.21*	− 0.14	− 0.10	− 0.13	0.03	− 0.05	0.38***	0.36***	0.46***	0.48***	0.39***	0.54***	0.59***

*p < 0.05; **p < 0.01; ***p < 0.001

Table 5 Multivariable analyses with food addiction score as the dependent variable (Nagelkerke $R^2 = 0.323$)

	Unstandardized Beta	Standardized Beta	*p*	95% CI
Age	0.45	0.26	**0.002**	0.17; 0.73
Psychological distress	0.63	0.35	**< 0.001**	0.34; 0.91
Cognitive reappraisal	− 0.17	− 0.14	0.114	− 0.38; 0.04
Cognitive self-consciousness	− 0.54	− 0.25	**0.006**	− 0.92; − 0.16

Numbers in bold indicate significant p values. Variables entered in the model are the ones that showed a $p < 0.002$ after Bonferroni correction for multiple testing

and clinical implications for alleviating FA symptoms in schizophrenia.

FA symptoms were found to be positively associated with patients' age. Consistently, findings from a systematic review showed that being aged over 35 years was correlated with an increased risk for FA [15]. However, mixed findings have also been reported. For instance, in two large studies involving exclusively female cohorts the prevalence of FA was inversely associated with age [51]. Similar patterns of negative association between FA and age were also found in male and female adults from the general population [73, 74]. Therefore, the specific impact of age on addictive food consumption remains unclear and poorly understood to date [66]. Finally, our findings could not confirm any significant association between psychotic symptoms and FA. Consistently, the existing body of literature clearly suggests that, in schizophrenia, overeating problems and excessive food consumption are considered to result from mood and cognitive symptoms [75, 76], whereas restrictive eating and limited food consumption are believed to be closely related to positive psychotic symptoms (hallucinations and delusions) [26, 51]. However, the relationship between psychotic symptoms and eating behaviors is still controversial. Although some findings reported more severe psychotic symptoms in patients with schizophrenia who had a history of an eating disorder [77], other ones noted clinical improvement in psychotic symptoms coinciding with higher food consumption [78], or, in contrast, a significant decrease in excessive food consumption as psychotic symptoms improved over time [79]. Finally, the lack of significant association between antipsychotics intake and FA in our sample concurs with previous research (e.g., [76]). In agreement with our result, disordered eating behaviors were observed in antipsychotic-naïve patients with schizophrenia [30], and were described even before the advent of antipsychotics [23], which lends more support to the role of psychopathology in the emergence of FA in this specific population.

Limitations

The current results should be interpreted with caution due to some limitations. The first limitation is inherent to the cross-sectional nature of our data, which did not allow for causal inferences to be made. Although a significant positive association was observed between psychological distress, functioning levels and FA in our study, it remains unclear whether FA is a cause or a result of psychopathology in patients with schizophrenia. Thus, future studies using experimental and longitudinal designs are required to explore whether these factors have a causal role in the development or exacerbation of FA symptoms in patients with schizophrenia. The lack of an accepted definition for FA represents another limitation. Besides, this study only relied on self-report measures to screen for FA symptoms. The mYFAS 2.0 used in this study is one of the most widely used and accepted tools for assessing FA in all populations, and has demonstrated excellent psychometric properties in its Arabic version [80]. However, future studies should consider using further assessments, such as structured clinical interviews, to detect individuals at high risk for eating disorders and those with existing eating disorder diagnoses. Furthermore, other important variables were not considered in the context of the present study and could have influenced the results. For instance, there is growing evidence linking impairments in executive function and greater severity of FA symptoms in various populations [81–83]. At the same time, it is well-established that executive dysfunction is a key psychopathological feature of schizophrenia that is caused by structural and functional brain abnormalities [84, 85]. Therefore, deficits in executive functioning could be a potential factor explaining the co-occurrence of schizophrenia and FA, and should be explored in future studies. Other important variables to be taken into account are body weight and other disordered eating problems, such as binge eating or bulimia nervosa. Because of a lack of resources in Tunisia, body weight measurement is poorly recorded in hospitalized patients, and could not, therefore, be gathered in the present study. Besides, due to time constraints and the desire to lower cognitive burden on respondents, other disordered eating measures were not included in the study's questionnaire. Future studies should consider including these relevant data.

Clinical implications and future research directions

Taken together, findings of this study hold some implications for the assessment and management of patients with new-onset schizophrenia. Overall, our results suggest that FA may be present soon after the diagnosis of schizophrenia, thus providing an important opportunity for prevention before overt complications (such as obesity or metabolic syndrome) occur and weight gain begins to accumulate under antipsychotic treatment. Along with monitoring weight and metabolic parameters, patients with first-episode schizophrenia who are newly established on an antipsychotic treatment should undergo routine monitoring of eating behaviors. The use of brief and easy-to-use screening tools, such as the mYFAS 2.0, can enhance rates of early identification and correct diagnosis, especially since this clinical entity is still not well known and recognized by clinicians [86]. An increase in early detection rates of FA in clinical care of patients with schizophrenia may help guide patient-centered treatment strategies, taking into account the neural and physiological mechanisms underpinning addiction to food, instead of current ineffective weight programs focusing on exercise and diet (e.g., [87]). Research on therapeutic interventions of FA is still in its early stage [16]. It was, for example, suggested that medications used to treat substance use disorders, such as bupropion and naltrexone, can help improve FA symptoms [88]. Moreover, glucagon-like peptide (GLP)-1 agonists appear to both have potential anti-addiction effects and diminish craving for food [89, 90]. In support to previous literature, our findings suggest that a specific type of metacognitive beliefs, i.e., cognitive self-consciousness, appears to be particularly relevant in patients with schizophrenia who present with FA symptoms. This could provide clinicians with a context for the emergence of FA symptoms and enable more tailored interventions. In particular, it is suggested that cognitive behavioral therapy aiming at modifying dysfunctional cognitions, the metacognition concerning cognitive self-consciousness in particular, can help prevent and manage FA in schizophrenia. Future experimental research is needed to confirm our findings and suggestions.

Conclusion

It is clear that the nature and correlates of FA in people diagnosed with schizophrenia are still insufficiently investigated and understood; we hope that this study has helped narrow this research gap. Our findings suggest that people with first-episode schizophrenia are likely to present with co-occurring FA, and experience associated distress and functioning impairments. This suggests that the identification and possible management of FA symptoms at early stages of schizophrenia could help alleviate distress, improve overall health of patients before further progress and complications like obesity occur. Future experimental research is needed to further explore the potential effects of treating FA on obesity and metabolic syndrome outcomes in people with early-stage schizophrenia.

Supplementary Information

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Additional file 1.

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FFR designed the study; YB processed the data; FFR and SH drafted the manuscript; SH carried out the analysis and interpreted the results; AH, WH and MC reviewed the paper for intellectual content; all authors reviewed the final manuscript and gave their consent.

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Availability of data and materials
The datasets generated and/or analyzed during the current study are not publicly available due to restrictions from the ethics committee but are available from the corresponding author on reasonable request.

Declarations

Ethics approval and consent to participate
Each participant provided a voluntary oral informed assent before beginning the survey. The research protocol was approved by the ethics committee of the Razi psychiatric hospital, Manouba, Tunisia (Reference number: ECRPH-2022-0076). The study was performed following the standards for medical research involving human subjects recommended by the Declaration of Helsinki for human research.

Consent for publication
Not applicable.

Competing interests
The authors declare that they have no competing interests.

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